THE CHIRONIAN.

VOL. XXVI JANUARY, 1910 No. 7

A METHOD OF FINDING THE SIMILIMUM IN DIFFICULT MIXED CASES.

By W. H. Freeman, M. D., Brooklyn, N. Y.

The following is an attempt to elucidate the method of symptom analysis grouping so essential, in many difficult cases, in order that the physician may sufficiently comprehend the symptoms that he will be capable of proceeding to the selection of a curative drug with a fair degree of success.

Case examination, as an homœopathic procedure, is divisible into three stages—to wit:—

First, the taking of the case or obtaining the totality of symptoms, which validly includes a correct diagnosis; a knowledge of all pathological lesions; and the time of existence of each symptom. Without such accuracy prescribing is more or less problematical.

Second, the analysis and proper grouping of symptoms, which forms the subject of this paper. By this second step in the process of case examination we are enabled to decide which are the important or ranking symptoms, always judged, of course, from the standpoint of the patient.

While in the majority of instances all that is necessary is the matching of a drug to all the symptoms of the case, such work is purely mechanical and will often fail in cases which are in any way complicated.

Only after having properly analyzed the symptoms will we be ready to proceed safely to the next and last stage.

Third, the selection of the similimum. Unless the prescriber is a master of homœopathic materia medica or is positively certain of the remedy by the time this stage has been reached, it is much better for him to consult the repertory, choosing the remedy by a process

of exclusion in some such manner as that about to be illustrated.

We all creep before learning to walk and the following is the method of reasoning by which essentials are differentiated from non-essentials in morbid symptomatology, somewhat difficult, perhaps, for the novice; but just as easy and matter of fact as walking when one has learned how. All that is required is a fair knowledge of physiology, pathology and diagnosis, together with ordinary common sense. The teaching of another to thoroughly master the science and art of Homœopathy is much more dicffiult than is the practice of such in itself; and procedures which may seem difficult and obstruse to the fainthearted beginner, become easy and are performed automatically after moderate experience and a mastery of the fundamental principles.

Not only is the method to be described the only logical one of studying cases but it is the method always pursued by the best prescribers, consciously or unconsciously, even though they are frequently unable to describe the process to others or account satisfactorily for certain of their curative prescription.

A demonstrated cure may be convincing and interesting but it is of no practical value to students or other practitioners unless the reasons for selecting the particular drug and the method of procedure are so clearly set forth that the observer can go and do likewise under similar circumstances.

As will be demonstrated by the following case, the similimum is not necesarily the drug covering the most symptoms, but rather the drug which best covers the peculiar and uncommon features of the case. Therefore, as has been already inferred, the selection of the curative drug will often depend entirely upon our ability to decide *which* are the important symptoms of the case.

The case to be presented being somewhat difficult and complicated, the analysis must, therefore, be somewhat extended; but it is hoped that the importance of the subject will justify the attention devoted to details.

The patient having been treated for several years by a good prescriber without benefit, it is necessary for us to study the case with exceptional care in order that we fail not also.

Though a minority of the following symptoms only are important, *e. g.*, uncommon and individualistic for such a case—all symptoms

will be briefly considered in order to clearly demonstrate, as far as possible, *why* some are important and others not so.

Were we to consider the important symptoms only, the object of the paper would be lost, because only one can choose a remedy for characteristic symptoms already picked out and arranged for him. The selection of the ranking symptoms often requires skill of the very highest order and it is such ability more than anything else which distinguishes the master from the apprentice.

Mrs. X, age 54.

X 1. Pain left side abdomen, paroxysmal.
X 2. — relieved by bending double.
X 3. — " " pressing fists into abdomen.
X 4. — " " lying on left side.
X 5. — worse standing or walking.
X 6. — urging to stool, with.
X 7. — relieved after stool.
X 8. — worse after onions, cabbage, fats, coffee.
X 9. — " " eggs.
X 10. — extending to lower dorsal vertebræ and left dorsal region.
X 11. — cramping, griping, colicky.
X 12. Stools variable, diarrhœa and constipation alternating.
X 13. — hard balls and lumps.
X 14. — fluid, offensive.
X 15. — mucus shreds in.
X 16. — gelatinous casts and mucus lumps in.
X 17. — excoriating.
X 18. — blood with a moderate degree.
X 19. Diarrhœa after eating eggs.
X 20. Has never used cathartics.
X 21. Frequent borborygmi with nausea.
X 22. " bloating better by kneading abdomen.
[24. Craving for bitter things all her life, as did her father also.]
X 25. Left inguinal pain—heavy bearing down feeling—worse standing or walking.
[26. Amenorrhœa for last five years.]
[27. Leucorrhœa worse standing or walking, off and on for many years.]

28. Cramps, calves during sleep.
29. Sleeplessness after excitement.
30. Numbness hands, morning on waking.
31. Urine, dark, concentrated, 1750 C.C.
32. — scalds on passing.
33. — retarded at times.
34. — indicanuria, moderate.
35. — solids and urea, absolute increase.
36. — albumin and sugar absent.
37. *Physical Examination:*—A tall, slender brunette of cheerful, vivacious disposition; heart, lungs, liver, and spleen negative; gastro-enteroptosis to a moderate degree; abdominal walls thin, lax, moderately potbellied—having had five pregnancies.

X 38. Painful sensitiveness on palpation over the descending colon and sigmoid region and over points of spinal origin of lower dorsal nerves on left side. No tumor palpable, no loss of flesh during illness. Examination of stool reveals characteristics already correctly described by the patient.

Diagnosis, chronic ulcerative colitis. The patient being unable to pay for it, a blood examination was not made.

By consulting the repertory it will be found that no one remedy covers all of the symptoms, therefore it becomes necessary to carefully analyze the case in order to decide which are most important—our own being to follow Hahnemann's advice and *"consider chiefly the more recent, most troublesome, and most peculiar or uncommon features"* to the exclusion of others not so classified.

We will begin, therefore, by eliminating and placing within brackets those symptoms of long standing which are not a part of the present ailment—to wit:—

Symptom No. 24:—Craving for bitter things all her life.

Symptom No. 26:—Amenorrhœa, which preceded the present ailment by several years and is obviously physiological for a woman of her age.

Symptom No. 27:—Leucorrhœa for many years, an old chronic symptom of mild degree.

It is now evident that most of the remaining symptoms are accountable for by the intestinal lesion; and that they can be classified either as *direct symptoms* (those directly associated with the diseased colon) or *indirect symptom* (those affecting other organs and functions secondarily, being due indirectly to mal functionating of the intestine).

Now, while it is seldom necessary to classify symptoms as direct or indirect, the value of a symptom usually depending solely upon its degree of peculiarty irrespective of such considerations as this, in this case the classification is necessary for the reason that *a course of now-curative-homœopathic-medication always produces more or less alteration of symptoms—new drug symptoms being added in place of natural disease symptoms removed or obscured.*

The important thing to remember in this connection is, that *it is chiefly the indirect symptoms which are changed;* the direct symptoms being altered least or not at all. Under such circumstances the direct symptoms will often be the only symptoms upon which to rely with any degree of certainty.

(While the diagnosis is *seldom* of value upon which to base the prescription, it is often essential as a starting point from which to begin the classification of symptoms. How otherwise can we decide, with any degree of safety, which symptoms are common and which uncommon, which indirect, etc., etc.? A common, low rank symptom of one disease may be a decidedly uncommon high rank symptom in another form of disease.)

We will, therefore, place an *asterisk* before every direct symptom, namely:—symptoms 1 to 23, 25, and 38; and placed to choose our ranking symptoms from those so designated, at the same time endeavoring to find the similimum by exclusion of drugs from corresponding rubrics in the repertory as we proceed:

*1. *Paroxysmal pain left abdomen.* Intestinal pains are usually paroxysmal; the descending colon is a left sided organ—therefore common and of low rank. Forty-two drugs have such pain and one hundred and fifty affect the left side of body.

*2. *Abdominal pain relieved by bending double.* While common to most colicky conditions, it is covered by comparatively few remedies; and clinical experience has proved it a valuable symptom in all abdominal conditions.

Kent's Repertory (1908 edition), page 557: Aloe, Ars. i., *Bell.*, Bov., Cast., *Caust., Chin.*, COLOC., *Colch., Cop.*, Cup., Euph., Eupi., *Iris*, KALI c., *Lach.*, Lyc., *Mag. p.*, Mang., Merc. c., Nux v., Petr., Phos., Podo., Prun., PULS., Senec., *Stann.*, Staph., Stram., Sul., Verb., Zinc.

*3, 4, 5. *Pain relieved by pressure of fists; lying on left side; and worse standing or walking*—should be translated to read *"painful and inflamed organ relieved by rest"* and common to the condition therefore. If the condition were purely neuralgic or functional, or if the modalities were the opposite, such would be uncommon and of high rank.

*6. *Urging to stool with the pain,* is common to the condition.

*7. *Pain relieved after stool,* can be explained on the basis of an irritating substance removed, though in many cases the pain continues or is worse. The symptom is covered by but few drugs of which by exclusion from the preceding drug list we obtain the following:—

P. 561: Aloe, Bov., COLOC., COLCH., NUX v., Senec., Sul.

*8. *Worse after onions, cabbage, fats, coffee.* Since the first three are digested in the lower intestine and the last is an intestinal stimulant, we would expect them to irritate the inflamed colon—therefore common modalities and of little value.

*9. *Worse after eggs.* Contrary to the last, eggs are

digested principally in the upper part of the digestive tract and give also but little excrement. This symptom being due to an idiosyncrasy and being uncommon to the pathological state assumes high rank.

P. 1324, Colch.

Kent gives but four drugs in this rubric, (Chin. a., Colch., Ferr., Ferr. m.); *Allen's Slip Repertory* gives in addition, Psor., Sanic., Selen., Tuberc.; and Sulphur is a drug which I have personally verified. *Colchicum* and *Sulphur* therefore remain for consideration.

*10. *Pain extending to lower dorsal vertebræ.* This is a mechanical low rank symptom, because irritation from an organ is prone to travel along the nerves and cause tenderness and pain in the neighborhood of the spinal center. On the other hand, when pain is reflected to a distant part via and away from the center, it is often a very important symptom, *because the point of reflection is subject to idiosyncrasy.*

*11. *Cramping and griping* are common to such conditions and to many drugs. While the similimum must be able to cause similar pains, such symptoms possess only a negative value from the differentiating or individualizing point of view because covered by so many drugs.

*12. *Diarrhœa and constipation alternating.* A peculiar symptom as worded and covered by Aloe, NUX, and *Sulphur* only, of the drugs previously considered. There is, however, a possibility of its being an irritation diarrhœa in an habitually constipated patient and therefore mechanical and common. A strong possibility that must not be overlooked is that *Aloe, Nux* and Sulphur have already been abused by the previous physician, such being the drugs that all homœopaths first think of for such conditions. Since Colchicum has both diarrhœa and constipation it will hardly be safe under the circumstances for us to rule it out in favor of Sulphur at this stage of the analysis.

*13. *Stools of hard balls and lumps.* Fairly com-

mon in such conditions and covered by *Nux* and SULPHUR only.

*14. *Stools offensive.* Common to condition. Aloe, *Colch.*, Nux, SUL.

*15. *Shreds of mucus in stool.* Covered by Colch., only.

*16. *Jelly-like mucus, lumps and casts.*

P. 637, *Aloe,* COLCH.

*17 and 18. *Stools bloody and excoriating. Aloe, Colch., Nux, Sul.*

*19. *Diarrhœa after eggs.* A very peculiar symptom and covered by but one remedy, Chin. a., but any one of the remedies in the general rubric which has been already considered (*9) is applicable for this symptom *and general rubrics should always be given preference to particular rubrics in choosing the remedy.* The remaining direct and indirect symptoms are more or less common and of secondary importance. In order to complete the analysis we will briefly consider them as follows:—*21, 22, 23. Are not covered by Colchicum as worded; but the drug has bloating, rumbling, belching, nausea and weakness to a marked degree.

*25. *Heavy bearing down. left inguinal, worse standing and waking.* Purely mechanical, the inflamed sigmoid would naturally be expected to feel so under the circumstances.

*38. The last of the direct symptoms. A pathological state of diagnostic value only because common to the condition.

Judged by the foregoing analysis Colchicum seems to possess the most reliable qualifications of any drug considered, although Sulphur is hard to rule out positively and it is highly probable that the latter will eventually be necessary to complete the cure.

Right here some may ask, such being the case, if it would not be well to give Sulphur at once and save time? The answer to this, however, is that Sulphur can never do the work of Colchicum or any other drug, nor will

it ever be of benefit when used out of its place or during the wrong stage of treatment. Also it has, without doubt, already been used without benefit because of the facts just stated.

The indirect symptoms will be briefly considered, as follows:—

*28, 29, 30 are individualistic and would be of value under other circumstances.

*28. *Cramps calves at night.* Nux, SUL.

*29. *Sleeplessness, excitement.* Colch., NUX, Sul.

*30. *Numbness hands.* Aloe, *Colch., Nux,* Sul.

*31 to 35. The urinary symptoms are common to the condition and to many drugs, including Colchicum and Sulphur.

*37. The physical examination, reveals nothing uncommon or characteristic. The fact that the patient is tall, slender, dark, and cheerful is of no importance except negatively—only when such is abnormal does it demand consideration.

July 28, 1908. ℞. *Colchicum,* 21 *m.,* one dose.

Aug. 5. Feeling much better. ℞. *Sac. Lac.*

Aug. 12. Not quite so well as last week. ℞. *Colch.* 21. *m.*

Aug. 21. Feeling splendid. ℞. *Sac. Lac.*

Aug. 31. Not so well for the last four or five days. ℞. *Colch.,* 21 *m.*

Sept. 9. At a standstill—not much relief from last medicine. ℞. *Colch. c.m.,* one dose.

Sept. 21. Symptoms have almost entirely disappeared since last visit. ℞. *Sac. Lac.*

Dec. 1. Entirely relieved since last visit. No medicine.

Jan. 11, 1909. A mild return of symptoms, this time calling unmistakably for and relieved by ℞. *Sulphur* c.m., one dose.

March 27. Another return of symptoms, this time calling for and quickly relieved by one dose of *Nux vomica c.m.*

July 21. Another slight return of symptoms quickly relieved by one dose of *Sulphur c.m.*

The potencies used in this case were not selected on account of any special favoritism for high attenuations but because in the judgment of the writer they would both correspond to the personality of the patient and the peculiar nature of the disease.

For a different personality and a different kind of case, the 200th; the 30th; the 6x; or even the θ might have been given.

In the opinion of the writer, it would be especially advantageous for our school, as a whole, if the special advocates of particular forms of potency would cease their bickerings and learn that all potencies have each their proper sphere and are necessary in order to obtain the best results in all cases.

While the majority of cases will do well under the right remedy in almost any form of potency, if judiciously used, a good many will not respond satisfactorily to potencies that are improperly selected.

THE DIAGNOSIS OF NERVOUS DIDEASES.

By John E. Wilson, M. D.

The condition confronting the practitioner of medicine was never so forcibly brought home to me as when a lady, a patient of mine when in general practice, asked me how it seemed to have ceased to profess omniscience. Most of you gentlemen must profess omniscience, and there is no need that I should come here to point out that such a position is fraught with more embarrassment every year as the fund of demonstrated fact becomes greater and greater. Omniscience demands frequent review of our fund of accumulated knowledge, and I have thought that it might be of mutual benefit if I ran over with you, even in the cursory manner necessitated by the limitations of time, the data by which we determine the presence and nature of some of the organic diseases of the nervous system.

Read before the Interstate Society, Oct., 1909.

As homœopathic physicians we are constantly impressed by the value of the totality of symptoms as the only guide to a successful prescription, and that "key-note" prescribing is not the highest development of the therapeutist's art. With equal truth we may affirm that one symptom, or even a triad of symptoms, will not infallibly guide us to right conclusions in the diagnosis of neurological conditions. Sometimes it seems as though any method was so hedged about by possibilities of error that any definite conclusion was out of the bounds of human attainment. It may, however, be affirmed that there are criteria which will infallibly narrow the extent of one's error, and very often altogether eliminate the possibility of it. The methods of which I shall speak involve the consideration of the presence or absence of a comparatively few physical conditions which can be ascertained without the volition of the patient, do not demand special apparatus beyond the easy attainment of any physician, and in many cases do not demand information from members of the family as to the order, rate and manner of evolution of the departure from the normal, although these data are vital to the diagnosis of a small proportion of cases.

The normal man walks and talks, he sees and hears, he drinks and defecates and urinates. He has intellect, and therefore he has desires and appreciations and dislikes. He has sexual organs and appetites, he has volitional impulses, and his members obey them. His tissues waste, and are repaired. He has organs of expression, and they reflect his changing emotions. From the perversion or abolition, partial or entire, of these we base our diagnosis of the presence of organic nervous disease, and localize the lesions by several criteria, principally by changes in those organic phenomena called reflexes. It has long been known that if one sharply strikes the tendon of a muscle in extension the muscle will at once contract, and that the occasion of this contraction has been ascribed to the motor response of the motor cells in the anterior horns of the spinal cord to a stimulation sent in to them from the sensory terminals in the tendon of the muscle. Since such a reflex occurs in .008 of a second, and the speed of transmission of nervous stimuli is only 30 meters per second, such a theory is manifestly attractive, but incorrect. The deduction that the loss of the response shows interruption of the nervous supply to the muscle is nevertheless cor-

rect. All muscles are capable of such a response, but most of them are so situated that they cannot be investigated with facility, and we are therefore limited to a rather small number, which, however, experience has shown to be sufficient for our needs.

These are the Achilles-jerk, which is a contraction of the gastrocnemius muscle from tapping the tendo-Achillis, and results in the extension of the foot into line with the leg. The knee-jerk is a contraction of the quadriceps extensor elicited by tapping the patellar tendon which causes the leg to be extended in line with the thigh. The radial-jerk is a contraction of the extensor carpi radialis extending the hand upon the forearm, and is brought about by tapping the tendon as it passes over the radius. The triceps-jerk is a contraction of the forearm and is caused by tapping the tendon of insertion. The jaw-jerk is a contraction of the masseter muscle which sharply brings up the lower jaw, and is developed by tapping the muscle over the lower edge of the zygoma. These reflexes are not always present to the same degree in different individuals, but a rough normal can be readily discovered, and they are very rarely totally absent in health (knee-jerk possibly absent in 1 per cent.). Exaggeration, if alike on both sides, has little organic significance as an isolated symptom, but inequality of the two sides, or great diminution or absence should always suggest organic nervous disease. The foregoing are tendon or deep reflexes. There are others of very great value.

The genital reflex is a contraction of the muscles of the abdomen upon squeezing the testicle: it is very constant, and its absence very significant. Stroking the skin on the inside aspect of the thigh will cause a retraction of the testicle on that side. Stroking the skin of the abdomen and of other parts of the body will cause retraction of the subjacent muscles, such reflexes being called skin, or cutaneous reflexes, and for comparison of the relative innervation of the two sides of the body are valuable, but are too inconstant in presence to be of equal diagnostic value with the tendon-reflexes. We must, however, make an exception in the case of the plantar reflex. If we stroke the sole of the foot of a healthy person, to a greater or less degree, the toes will be sharply flexed upon the sole. If in response to such stimulation, which must be carried to the point of pain before abandoning the attempt, we find that instead of being

flexed upon the sole of the foot, the toes, perhaps the great toe only with the same significance, are sharply over-extended upon the dorsum of the foot. This is Babinski's reflex, and is a certain sign of organic disease, but its absence does not prove the absence of organic lesion. Stroking the skin on the inner aspect of the tibia is said to provoke the same response, and is called Oppenheim's reflex.

Another sort of reflex is called clonus, and is equally indicative of organic disease if persistent. If one grasps the toes, and firmly by them pushes the foot toward the knee the over-straining of the calf-muscles irritates them, if organically diseased, and the foot will oscillate to and fro. This is called ankle-clonus. Sometimes it may be exhibited in the patella by pushing downward upon it. If we place a tongue depressor or plessimeter on the teeth of the slightly depressed lower jaw, we may get a similar response with the same significance as to the condition of the masticatory muscles. This is jaw-clonus, and in passing I will note that it is found only in cerebral lesions, and amyotrophic lateral sclerosis.

If we percuss a muscle of a healthy person it will contract as a whole with a marked elevating at the point of percussion. If, when we percuss it, we see a worm-like contraction of single fibres only, and the sum of all these contractions is not sufficient to move the limb, we have developed fibrillation, which is always the sign of organic disease in the cell-nuclei of the anterior horns of the spinal cord. A sudden exposure of the part to cold may be sufficient to produce it.

In health our skin sensations keep us informed of sensory impressions of all kinds, but by separate mechanisms for the various forms of sensory impression: touch, pain, heat, and cold have separate recipient organs, and to some extent separate tracts, and recipient cells. When we find these perceptions exaggerated, diminished, or absent, we have positive information that the nervous condition of the person is perverted, although that alone does not give us warrant to affirm that it is the result of organic disease. Two varieties of loss are, however, certain indications of this. If we can appreciate touch sensations, and fail to appreciate pain sensations and changes of temperature, we know that the cells about the central canal of the cord. or the tracts leading to them, are destroyed.

Secondly: we maintain our station, and recognize the position of our limbs by sensations coming by special fibres from our tendons and joint-surfaces. If we lose this sense we are ataxic, and these fibres, or the cells in the cord to which they are tending, or the tract on the lateral portion of the cord, or the cerebellum, to which they are going, are certainly diseased.

We all know that waste and repair are the cycle of life. If emaciation is general, we look for a general cause; but if it is local, our source must be more specific. Localized atrophies, and hypertrophies, as well, are indication of organic nervous disease.

The foregoing are the criteria by which we can establish a diagnosis of nervous disease, but to certainly group these discovered and localized lesions into a symptom-complex, as we must often do for the purpose of prognosis, treatment, and description, we may need a knowledge of the method of onset, and the order and rapidity of development of these symptoms. Our data then are compiled from the inspection of the mode of locomotion, the efficiency of the arms and legs, the eyes, ears, lips, mouth, and the tongue; the perfection of swallowing, and of speech, of vision, and of hearing, and of smell, since for diagnostic purposes they may all be considered motor phenomena. Then comes the investigation of the reflexes, both tendon and cutaneous, including touch, pain, and temperature perceptions; the presence of fibrillation, of atrophy or hypertrophy, and of ataxia. The investigation of the reflexes may follow cursory inspection, since by them alone we are able at once to divide almost all diseases into those of either the upper or lower motor neuron, and thus cut the field of necessary investigation in half.

The motor nerve-path running from the motor cell in the cortex of the brain to the end-plate in a given muscle may be considered to be divided into two distinct parts. The cortical cell in the brain, and its fibre, including that portion where it breaks up into an end-brush by which it influences the motor cell in the anterior horn of the spinal cord is in its entirety the upper motor neuron. The motor cell in the cord, with its fibre to the muscle, and its end-plate in the muscle, make up the lower neuron, and lesions of either neuron have distinctive characteristics. We can assume with certainty that if the upper neuron is injured in any part the tendon-reflexes in the parts below will be exaggerated, (unless the whole cord is

absolutely divided), that atrophy will be moderate and slow, and proportioned to the diminution in the use of the parts, that muscles will probably react to faradism, and certainly more vigorously to the galvanic cathodal closure than to the anodal. If the lesion is in the lower motor neuron the tendon-reflexes will be reduced or abolished, atrophy will be prompt and excessive, and select certain muscles, the reaction to faradism will be speedily lost, while the muscles will react equally, or more readily to the anodal galvanic closure than to the cathodal. This is the reaction of degeneration.

Localization of the paralysis is another aid to diagnosis. Suppose that we have decided that a given case is the result of an upper neuron lesion, we still have to decide whether the cerebral or spinal portion of the neuron is affected. Localization of the paralysis will be hemiplegic, not necessarily the whole of one side, but only wholly upon one side, if the lesion is cerebral; while a spinal lesion of the upper neuron type produces a paraplegia. A cerebral paraplegia is humanly possible, a diplegia is not very rare, but the history of such cases will show that they are always the result of two apoplectiform attacks, appreciably separated in time, making the condition actually a double hemiplegia.

A lower neuron paralysis may be the result of a lesion of the lower motor neuron in any one of two situations, *i. e.*, in the cells in the anterior horn of the cord, or in the nerve in continuity, sometimes divided into segmental, the paralysis will be devoid of sensory symptoms, except of a very slight nature, almost purely motor, and selecting the muscles according to their representation in the affected segments of the cord. Wasting will be prompt, excessive, and selective, and marked by fibrillation, and there will be the reaction of degeneration. If the lesion is peripheral the muscles affected by paralysis will be such as are known to be innervated from definite nerve-trunks, or a single nerve, the paralysis will be marked by the presence of sensory symptoms, except in the rare cases of purely motor branches, there will be wasting, but more gradual than in segmental lesions, the reaction of degeneration, and definite skin areas will show paresthesias followed by anesthesia.

Recall that the division into upper and lower neuron lesions relates only to lesions of the motor tracts. We are not, however, left helpless in lesions of other areas, for their functions are known.

The front areas of the cord are purely motor and trophic, and if a spinal paralysis is characterized solely by motor and trophic symptoms we know the lesion to be in this area. The lateral area of the cord is occupied by the motor tracts from the brain, and the direct tract to the cerebellum. A lesion here will give us an upper neuron type of paralysis, with the addition of ataxia. The posterior part of the cord is purely sensory, and lesions of that part will, of course, produce sensory, or sensory and ataxic symptoms only. These sensory lesions may be in the cord, or be peripheral. If one or more limbs be the seat of an anesthetic like a glove or stocking, up to a certain level, we know that we are seeing the results of a peripheral nerve-lesion, hysteria being eliminated. If, on the contrary, the anesthesia is in scattered areas here and there over the body, it is the result of a lesion of the sensory tract in the cord or brain. If both sides of the body are insensitive below a certain level, at that line are over-sensitive, and above it are normal, we know that a little above the line of sensitiveness is located a lesion of the whole cord, motor symptoms betraying any extension into other areas. If one side of the body is insensitive below a certain line, and if at the top of this area there is a band of sensitiveness on the opposite side of the body, and if above this latter band the entire body is normal, we know that there has been a transverse lesion of one-half the cord on the side where the anesthesia was in a narrow band. Brown-Sequard type of paralysis.

From this resumé it will be gathered that lesions in the sensory tracts cannot usually be as accurately localized as in the motor tracts, but I hope that it has been made evident that a combined study of the general appearance of the person followed by an investigation of the condition of the reflexes, both tendon and cutaneous, of the localization of the paralysis, and also of the causative lesions, will enable one to diagnose most organic diseases, although they are not always purely of one or the other neuron or portion of the nervous system. Let us now put the foregoing assumption to the test, remembering that the great bar to correct diagnosis is the habit of basing diagnosis upon the presence of one symptom, like ataxia, for instance, and then beginning a hunt for confirmatory symptoms. The only proper method is to find first the exact condition of the patient in all respects.

Suppose that we have a patient with a spastic paralysis of the lower limbs, rigid in all movements, and somewhat the same condition in the upper limbs also, with no sensory symptoms, so far as we can see. The disease may be lateral sclerosis, ataxic paraplegia, multiple sclerosis, amyotrophic lateral sclerosis, syringomyelia, tumor of the cord, spinal hæmorrhage, or Pott's disease. Lateral sclerosis may be taken as the typically pure lesion of the motor tracts lying laterally in the cord, showing only rigidity, weakness, increased deep reflexes, perhaps clonus, and Babinski, but no sensory or associated symptoms. If now we should find that our anamnesis (information collected by examination), revealed the presence also of inco-ordination of movement, and a marked exhibition of it in an ataxic gait and station, we should be certain that the direct cerebellar tract was also involved, and that the condition was one of ataxic paraplegia. If the inco-ordination was of the upper limbs, with an intention tremor of the arms, scanning speech, and nystagmus, we could be reasonably certain that a spastic paraplegia with these associated symptoms was multiple sclerosis. If, on the contrary, there had been discovered with the symptoms of a spastic paraplegia a preliminary spastic paralysis of the arms followed by a more or less selective wasting, and possibly difficulties of deglutition amounting almost to suffocation, we could be sure that the cell-nuclei in the anterior horns of the cord and of the bulb were also involved, and that the disease was amyotrophic lateral clerosis. Let us suppose that the patient showed spastic paralysis of the lower limbs, and that the upper limbs remind us of amyotrophic lateral sclerosis, but with the distinction that the atrophy was of a most intense kind, leading to whitlow on several of the fingers, yet without pain, perhaps accompanied by a marked claw-like contraction of the hands and some lateral curvature of the spine. This would show us that there was a lesion in the cells of the anterior horns. The question might then occur to us whether this also was a case of amyotrophic lateral sclerosis. Our anamnecis would, however, show us that the patient, while sensible of touch, was insensitive to pain and changes of temperature, and that would infallibly point to the extension of the lesion, to the group of cells about the central canal of the cord, and to the identification of the disease as syringomyelia.

To take another case. A patient might show a spastic paralysis of the lower limbs, but in addition some diminution or sensation, or change of it in the lower limbs, with a tenderness of the spine and pain along the course of the spinal nerves, a voluntary rigidity of carriage, and a disinclination to passive movements of the spine. The cause would be Pott's disease, if we could discover some systemic disturbance, palpable angular distortion of the spine, or necrosis, by the X-ray. It might be tumor of the spine or cord to be diagnosed from the foregoing mainly by exclusion, or a spinal hæmorrhage which would be indicated by an apoplectic suddenness of onset.

Let us now take up the numerous diseases, which we exclude as evidently of the lower neuron type.

If a patient should present himself with a glove or stocking anesthesia of both fore-arms, or both legs, or all four extremities, a paralysis with wasting of the muscles, with lost reflexes in these areas, and the reaction of degeneration, we should certainly be in the presence of a widely disseminated paralysis of the lower neuron type. Can we learn more? If it was from a lesion in the spinal cord, of a general distribution, there would be spastic symptoms somewhere. If exclusively in the cells of the anterior horns there would be no sensory symptoms. The lesion must, therefore, lie outside the spinal cord, *i. e.,* be peripheral. Is the lesion in the nerve at its exit from the cord? If such were the case there would be some areas of exaggerated sensibility on the upper parts of the limbs in question. It is then not only peripheral, but the distal part of the peripheral nerve-trunk. Peripheral and what else? Both arms, or both legs, or all four extremities are affected, therefore it is symmetrical. Multiple neuritis is the only peripheral, symmetrical, acute paralytic nervous disease of which we have knowledge.

Another patient presents himself, who was in good health only a a few days ago, and yet, after a short feverish attack, finds himself paralyzed in one leg in a peculiar manner, (it would be just as true of an arm, but not as usual). After a few days of general paralysis of his leg, he finds that while some of the muscles are normal, others are so wasted that they seem to have been scooped out, and are utterly powerless. So far as these muscles can affect them the re-

flexes are lost; there is the reaction of degeneration, the limb is cold, but sensation is hardly disturbed beyond a little crampy pain confined to the affected muscles. It is evidently a lower neuron paralysis, but where is that neuron? No sensory changes, so the nerve cannot be the primary site of attack. Particular groups of muscles are represented in definite segments of the cord, and a study of the paralyzed muscles in this light will show that the muscles innervated from one, two, or three (generally) contiguous segments of the cord would innervate all the muscles which are paralyzed. Trophic and motor impulses are centered alike in the anterior horns, and we noted the simultaneousness of the paralysis and atrophy. The atrophy, in other words, is primary, and not from disuse. These facts stamp the paralysis as nuclear, and the fact that it was acute bars out hæmorrhage, which would be instantaneous, and degeneration, which would be chronic in its evolution. The only diesase which would conform to these criteria is anterior poliomyelitis acuta. If the process were chronic it would be some form of progressive muscular atrophy.

Let us now investigate the applicability of our method to a more complicated problem, one in which all varieties of nervous structure are involved.

If to the picture of a lateral sclerosis as just given, with paralysis of the lower limbs of an extreme grade, to which were added incontinence of urine, rapid wasting of the limbs, and tissue destruction even to the production of bedsores, with a band of tenderness and pain about the trunk, below which was insensitiveness, we should appreciate that it was an upper neuron paralysis with some novel peculiarities. The increase of the deep reflexes would indicate a lesion of the motor tracts in the upper neuron, the paralysis with rigidity would indicate the same, the rapid wasting would indicate a lesion of the anterior horn cells, and the absence of sensation in all portions of the body below a certain level would show that the sensory tracts were cut off, and since this condition is bilateral it would indicate a bilateral lesion. A transverse lesion of the cord just below the zone of hyperesthesia is the only lesion which would be competent to produce this condition, and this indicates a transverse myelitis.

Suppose that a person showed a definite helplessness in the lower

limbs without actual paralysis, loss of knee-jerks, and some changes of sensation in the lower limbs, with urinary incontinence, and we could obtain no history. The patient cannot walk well, but we find the muscles are as strong as ever, and the condition is evidently not a paralysis. The knee-jerk is absent, while the quadriceps extensor is as strong as ever. It is evidently a purely sensory lesion. It cannot be due to a lesion of the spinal nerve, since that would give motor paralysis in a definite area of the limb, and sensory loss as well, specifically in the area of distribution of certain nerves. Movements are inco-ordinate, and not paralytic, sensation is paralyzed in a somewhat selective way, but erratic in localization, causing in certain areas the impression of pressure from stimulation which should have produced pain, or lost sensation here, and exaggeration there. Ataxia and such selective sensory losses indicate a lesion of the fibres of the posterior portion of the cord. Such a condition would make the patient sway in station, and be uncertain in gait. He certainly would give us a strong foundation for a diagnosis of tabes dorsalis, and if we found that he had an Argyll-Robertson pupil, with a possible ptosis, or diplopia, we could be certain of it.

In the foregiong pages the author has endeavored to show the pathological basis for and the diagnostic value of the various phenomena discoverable in the subject suffering from organic nervous disease, and in conclusion would present the following recapitulation. All paralyses can be divided into those of cerebral or spinal origin, and then again be sub-divided into those of either the upper or lower motor neuron. The upper can again be sub-divided into those of the brain or of the spinal cord, and these again can be reduced to a very small group for final investigation by the decision of their hæmorrhagic, inflammatory, or degenerative character. Those of the lower motor neuron may, with equal facility, be grouped as spinal or peripheral, and the resulting small group may again be reduced in size by the classification into hæmorrhagic, inflammatory, or degenerative as above. When we have finally decided that the lesion is spinal we can finally and definitely identify it from our knowledge of the specific functions of the various tracts of ganglia of the cord, nd peripheral diseases betray their pathology by, first, of the character of the losses entailed by the disease, and secondly, by the distribution of these losses.

616 Madison Ave., New York City, Nov. 26th, 1909.

THE TOXIC EFFECTS OF ADRENALIZATION OF THE NASAL MUCOUS MEMBRANE.

By Marcel Lermoyez and Charles Aubertin.

(Translated form *Annales des Malades de L'Oreille, du Larynx, du Nez, et du Pharynx,* September, 1909. By Walter Sands Mills, A. B., M. D.)

I.

Adrenalin was discovered in 1901, in New York, by Jokichi Takamine.[1]

After the first analyses had been made by J. B. Moore,[2] in 1894, the active principles of the suprarenal glands was actively sought. J. J. Abel[3] believed he had found it in "epinephrine" in 1897; Von Furth[4] declared this to be an impure and almost inert body, and put forward a dioxypyridine hydrogenee, which he named "suprarenine," in 1900.

Takamine then discovered a third body, until then unknown, which he called "adrenalin," which is today considered the active principle of the medullary substance of the suprarenal glands.

Physiological Effects. The physiological effects of adrenalin are exerted only on the circulatory system.

A. *Local Action.* The local action is first an intense vascular constriction,[5] which, in consequence, causes an immediate anemia of the mucous membrane (like porcelain); this action is useful in the surgery of rhinology.[6]

This vascular constriction is purely of peripheral origin. The vascular spasm is produced without the intervention of the vasomotor centers. Joshué has been able to obtain a complete vascular constriction, a little retarded, it is true, in the ear of a rabbit after cutting of the cervical ganglion above the sympathetic.[7]

Thus, then, the anemia of adrenalin is first of all a local phenomenon; in the beginning of the organism is not affected as a whole.

B. *General Action.* The second effect of adrenalin, the general effect, does not follow except after intra-venous injection, it is an *elevation of the arterial* tension. Thus, Takamine noted that the intra-venous injection of a millionth of a gram of adrenalin per kilogram of animal elevated the blood pressure 14 millimeters of mercury.

This hypertension is the result, (a) of the local action of the adrenalin on the muscular element of the arterioles; (b) of the increased energy of the cardiac systole by direct excitation of the myocardium; (c) perhaps of an associated action on the nerve centers (Cushing-Reichert).[8]

Arising from this are two therapeutic corollaries:

a. The danger of adrenalin in diseases where arterial tension is raised, or when it is supposed that the cerebral arteries are deteriorated, or when there exists an arterial aneurism.

b. The advantage of adrenalin in cardiac asthenia following toxic infections, as a heroic measure to revive a failing myocardium whether in the collapse of fevers,[9] and in particular, in diphtheria (netter), or in heart failure from anesthesia by chloroform.

Toxic Effects. Adrenalin is extremely poisonous, as are all active principles of the secretions of internal glands. It is curious that Takamine did not recognize this toxicity.

Adrenalin produces, according to the way in which it is administered, an *acute intoxication,* or a *chronic intoxication.*

A. *Acute Poisoning.* To a slight degree there are nausea, vomiting, vertigo, a sensation of thoracic constriction with anguish, like the syndrome of angina pectoris.[10]

In a more intense degree, it has been observed especially in experiments on animals, that adrenalin intoxication kills very rapidly by acute *pulmonary edema.*[11]

In man, the syndrome produced by adrenalization is analogous to the typical acute pulmonary edema of arteriosclerosis; this has led Joshué[12] to state that in these cases pulmonary edema is caused by an exaggerated activity of the supra-renal glands.

B. Chronic Poisoning. This is manifested in two ways:—by arterial atheroma and by hypertrophy of the heart.

a. Slow poisoning by adrenalin (by way of the veins) produces *arterial atheroma experimentally,* without previously vascular traumatism being necessary. This remarkable discovery of Josné[13] has been confirmed by all experiments made since to verify it. At the autopsy on a rabbit which had received every other day an injection into a vein of the ear of three drops of a solution of adrenalin one to a thousand, there were found infiltrated spots in the aorta of an atheromatous type.

Adrenalin acts on the arteries as a local poison. The atheromatous effect is independent of the hypertension. Degrez and Chevalier[14] have shown that choline, injected into the vein at the same time, as adenalin, neutralizes the hypertensive effect of the latter. But, according to the experiences of Teissier and Thévenot,[15] the addition of choline to adrenalin does not prevent the production of the athermatous lesions; these vascular changes are as marked as when adrenalin is used alone.

The experiments of Josné do not confirm that, as those who believe in the theory that hypertension is always the origin of arteriosclerosis and atheroma may, perhaps, expect.

b. Slow poisoning by adrenalin also produces hypertrophy of the heart (o. Josné)[16] simple hypertrophy without degeneration of the muscular fiber.

This is due to the same local toxic effect as arterial atheroma; but this is of no consequence, for it is possible to produce it experimentally in the absence of all atheromatous formation.

From these beautiful experiments evidently springs the conclusion, that the totality of accidents one sees in atheroma seem to depend on a super-activity of the suprarenal capsules, which pour into the circulation a very large quantity of adrenalin.

Variations in the Toxic Effects. The effects of adrenalin poisoning are multiple. They vary (a) according to the dose of adrenalin; (b) according to the susceptibility of the individual; (c) according to the method of introduction of the poison.

a. Variation in the toxicity according to the dose of adrenalin:— Josné, in poducing experimental atheroma without determining an immediate prejudicial effect, ingested into a vein of the ear of a rabbit of 2 kilo. weight, three drops of a solution of 1 to 1000. But four drops were badly supported and killed in a few moments the same animal by pulmonary edema.[17]

Bouchard and Claude, according to Lépine,[18] state the fatal dose to be two-tenths of a milligram per kilogram of animal weight in intravenous injection. Nevetheless the habit develops so quickly that a dose of four-tenths is soon supported.

On the other hand, an important fact to rememebr in rhinological surgery—adrenalin has no cumulative effect, due, without doubt, to rapid oxidation in the organism (Batelli).[19] The action is produced very rapidly, but ceases just as quickly.

b. Variations in the toxicity according to the susceptibility of the individual. Man is infinitely more susceptible to the action of adrenalin than are the animals of the laboratory. It would be a mistake then to establish a posology for man according to experiments made on animals.

This difference is shown more especially when adrenalin is introduced hypodermically. Both Batelli and Josné, after a dose of 4 milligrams of adrenalin, so injected, failed to find any appreciable results.

In man—healthy individuals, tubercular individuals, etc.—Sonques and Morel have seen serious and dangerous syncope follow the sub-cutaneous injection of three-quarters of a milligram of adrenalin.

This susceptibility is truly remarkable and should hold one back.

On the other hand, intolerance to adrenalin is not constant; it is sometimes exaggerated.

A patient of O'Konneff[20] exhibited symptoms of grave poisoning (violent palpitations, vomiting, etc.) after dusting the throat with a weak 1-10,000 preparation of adrenalin. Sometimes morphine or quinine are likewise poisonous in infinitisimal doses.

Apropos of this, Josné noted abrupt changes in the toxicity of adrenalin that surprised him, without being able to anticipate in any way whether more or less would produce them. Thus one rabbit, perhaps, would succumb in a few minutes to pulmonary edema after the intravenous injection of a dose perfectly tolerated the day before. On the other hand, another animal would support one day a dose that previously had caused dangerous symptoms. One needs, then, to be on guard, from the therapeutic standpoint, against the lack of constancy in the toxicity of solutions of adrenalin. Such things are not surprising, for the experiments of Batelli have shown with what ease adrenalin is oxidized by light, even in a closed vessel, and as oxidized adrenalin is inactive, the result is a great variation in the effects. Pink, the solutions seem more active; dark they become almost inert.

Nothing is more important than to repeat the need of being on one's guard in the use of adrenalin in arteriosclerosis. Frey[21] has published a persistant case of hemiplegia following an operation by Caldwell-Luc made under local anesthesia by coca-adrenalin.

C. *Variations in the toxicity according to the method of ad-*

ministration of adrenalin. The toxic effects of adrenalin vary considerably according to the way of intoxication of this substance into the oganism.

In this regard it is necessary to distinguish:

1. Intravenous or intrapulmonary injection.
2. Intratracheal injection.
3. Sub-cutaneous or sub-mucous injection.
4. Ingestion by way of the digestive tract.
5. Application to mucous membranes.

Much more than the question of dose, the manner of using adrenalin interests the rhinologist.

1. *Intravenous injection.* It is when injected into the veins that adrenalin shows the most toxicity.

a. Hypertension is produced in a few seconds; the pressure rises rapidly from 12 to 23 centimeters of mercury, to fall again very quickly to below normal (done experiemntally and clinically).

b. The onset of acute edema of the lungs is caused by this method with terrible rapidity; ordinarily it kills the animal in a few hours (extreme dyspnea with expectoration of red froth).

c. Lesions of atheroma of the aorta and of hypertrophy of the heart develop in about a month's time, after ten or so intravenous injections have been made every other day (Josné).

The toxic effects of adrenalin are also marked when injected directly into the parenchyma of the lung, as has been proposed to do in man for the teatment of hemoptysis.

2. *Intratracheal injection.* Adrenalin administered by this method is as dangerous as in the preceding case. The possible effect should be considered when one would combat an hemoptysis in this way, or when one would perform an operation by way of the tracheobronchoscope.

Kulbs (cited by Josné) introduced for two consecutive days one-quarter of a milligram of adrenalin into the trachea of a rabbit. He saw the animal succumb with fulminating edema of the lungs two minutes after the second injection.

Another rabbit that received a twentieth of a milligram daily in the trachea, died in the same way on the seventh day.

The extreme and rapid absorption of medicines introduced by way of the trachea makes it easy to understand the gravity of these accidents.

3. *Sub-cutaneous injection.* What is to be said apropos of this mode of administration of adrenalin applies also to sub-mucous injection, actually much in favor in surgical rhinology.

Hypodermic injections of adrenalin are less efficacious, but also less dangerous than intravenous injections.

They are less efficacious, but not ineffectual as has been claimed. Sonques and Morel have observed arterial tension to be raised five centimeters of mercury in man, particularly in tubercular hemoptysis. Labbé and Amenille have also observed, in a parallel case, an intense peripheral vaso-constriction. On the other hand, hemiplegia supervened in a patient of Frey, mentioned above, testifying to its activity. Josné has utilized the action of sub-cutaneous injections of adrenalin to stimulate the energies of a failing myocardium in typhoid.

They are less dangerous. Thus, to cause fatal pulmonary edema, it was necessary to inject under the skin of a rabbit weighing 2 kilograms, 5 cubic centimeters of a 1-1,000 solution of adrenalin (Josné), when ten drops ingested into the veins almost immediately caused death.

On the other hand, Josné and the majority of experimenters have never seen arterial atheroma follow injections of adrenalin under the skin.

One may be authorized, in man, to use sub-cutaneous or sub-mucous injections of adrenalin in a dose of one milligram in twenty-four hours, without danger of pulmonary edema. But this should not be continued for more than ten days (Josné),[22] for fear of producing cardiac hypertrophy.[23]

4. *Ingestion by way of the digestive tract.* The toxicity of adrenalin is infinitely less when administered by way of the mouth.

According to Josné, one may prescribe without inconvenience, in man, 2 milligrams of adrenalin a day, by ingestion.[24]

The experiences of this author show that one may exceed this dose. He introduced into the small intestine of a rabbit, by means of an abdominal incision, as much as 3 milligrams of adrenalin per kilogram of animal, without observing any toxic phenomena. And he has never been able to produce experimental lesions of the arteries when using this form of administration. A strong reason why this dose should remain innocuous is that it is mixed with the

food, and, consequently, is more slowly absorbed. Thus Netter employed 4 milligrams in patients, without producing toxic effects, by dividing this dose into six parts during twenty-four hours.

The slight activity of adrenalin when thus administered is explained by the antitoxic action of the liver. This neutralizing effect of the hepatic cell has been elegantly demonstrated in the description by Carnot and Jusserand,[25] who stated that adrenalin elevated arterial tension less when injected into a mesenteric vein than when injected into a peripheral vein.

The relative innocuousness of adrenalin when introduced into the digestive tube merits the interest of rhinologists. Whatever they have to fear as to the entrance into the body by way of the inferior air passages, there is little to fear when the patient accidentally swallows a part of the adrenalin solution introduced into the nasal fossa.

5. *Application to mucous membranes.* Up to this point we have directed attention particularly to the authorities in this work.

It is on this method that we have conducted personal researches.

Josné has been led, by his experiments, to conclude that the innocuousness of adrenalin placed in contact with the nasal mucous membrane (applied on the surface) remains the same in experimental doses infinitely more strong than those which are employed off hand by rhinologists in order to obtain bloodlessness of the Schneiderian membrane before operation.

Following this author: 1st. Acute pulmonary edema is not to be feared; he has injected one cubic centimeter of a solution of 1-1,000 in each nostril of a rabbit of two kilograms, without seeing the least symptom of poisoning. 2d. Chronic vascular lesions have never followed. Josné, apropos of this, has written: "I have injected for a long time large quantities of adrenalin into the nostrils of rabbits. The first rabbit had eighteen instillations in fifty days, and received altogether 6.25 milligrams of adrenalin. A second rabbit, in sixty days, received 10.45 milligrams of adrenalin. Neither of these animals presented any vascular lesion.

Such an affirmation should not encourage rhinologists to abuse the use of adrenalin in nasal affliction. We believe it better to guard against doing this, for our personal experience, while in part confirming those of Josné, show that there may be danger in it.

(*To be continued.*)

EDITORIAL

THE GRANULAR FORM OF THE TUBERCLE BACILLUS.

For many years it has been known and commented on, that in many cases of pulmonary tuberculosis it was not possible to demonstrate the tubercle bacillus, or in other cases they would, at times, be plentiful, to be followed by periods during which they would disappear.

Surgeons and pathologists have long sought an explanation of the fact that in undoubted tubercular abscesses and ulcers the tubercle bacillus so often would be missing and yet the contents of such abscesses would produce a typical tuberculosis in the guinea pig.

In March, 1909, Much gave the answer to these questions by the announcement that there is a form of tubercle bacillus that does not stain by the ordinary Ziehl method but does stain by the Gram method. These were called the granular forms and were regarded as diagnostic. He recognizes three forms of the bacillus.

1. Bacilli disintegrated in granules.
2. Very delicate rods, stained only by Gram.
3. Acid-fast bacilli impregnated by fatty-acid substances.

In reviewing the work of Much, Liebermeister, of Koln, points out that such granules are found not only in tubercle bacilli but in all other bacteria. These granules are remarkably resistant to all chemical and physical influences. They are recognized in many bacterial preparations by darkening the field and in young tubercle bacilli even in unstained preparations. They stain pale orange or rose red with Sudan III.; give the naphthol blue reaction but do not blacken with osmic acid. They are not extracted with alcohol, ether, chloroform or benzine and resist for a long time digestion with pepsin, trypsin and papayotin. They are easily demonstrable in many kinds of bacteria with various staining methods.

The chief practical importance of Much's studies are the facts that tubercle bacilli and their granules stain with Gram after they are no longer demonstrable with Ziehl; that is, the bacilli can lose their Ziehl staining and wax-like coating and yet the bacilli and their granules remain undestroyed. The granules are so resistant to chemical and physical influence that they have been found intact after the bacilli have disintegrated.

OBITUARY.

Frank E. Risley, M. D., '89.

Frank E. Risley, M. D., died at 164 Herkimer street, Brooklyn, N. Y., Friday morning, December 10th. Dr. Risley was a graduate of the New York Homœopathic Medical College, class of 1889. He practiced medicine in Brooklyn for twenty years, with the exception of the three years in which he conducted the Rivercliff Sanitarium, at New Milford, Conn. He had endeared himself to a large clientele by his uniformly kind, gentle and sympathetic nature. For fifteen years he was physician-in-charge of the Brooklyn Nursery and Infant Hospital on Herkimer street, where he gave much of his valuable time in daily service. He was also a member of the visiting staff of Cumberland Street and Prospect Heights Hospitals. He was a member of the Kings County Homœopathic Medical Society, and for many years an active member of the Chiron, the Brooklyn Medical and the Inter-Nos Medical Clubs. He was a member of Philadelphos Council, Royal Arcanum, and Kilwinning Lodge, F. and A. M. He was unmarried, and leaves a sister and a brother. Interment will be made at his former home in Waterville, N. Y.

Hugh S. Blakeney.

Hugh S. Blakeney, of the Senior Class of the New York Homœopathic Medical College and Flower Hospital, met with a fatal accident on December nineteenth, while on his way home to Paris, Texas, to spend the Christmas holidays. To his class-mates and friends his death is a severe loss; he will be remembered as a loyal supporter of every college interest. The sympathy of the Class was extended to his parents in the following resolution:

WHEREAS, It has pleased God in His Providence to take from our midst our Class-mate, Hugh S. Blakeney, we, the Class of 1910, hereby extend to his parents our heartfelt sympathy and condolence in their profound bereavement.

We deeply feel the loss of one earnest, true, and much beloved companion, whose memory will ever be held dear to us."

CLASS OF 1910.

ALUMNI NOTES.

William McLean, M. D., '06, announces the removal of his office December the tenth, to 30 East 55th Street, Cor. Madison Avenue. Telephone, 6967 Plaza. Hours, Mon., Wed. and Fri., 1 to 2:30 P. M.; Tues., Thurs. and Sat., 1 to 1:30 P. M. Disease of the eye and ear.

Dr. Royal S. Copeland offers his services to the profession for operative and consultation work in the medical and surgical diseases of the eye. He holds himself in readiness to answer calls for this purpose in city or country. Patients will be seen for consultation, refraction, or treatment during the regular office hours or, preferably, by appointment. Hours, 10 A. M. to 1 P. M., except Sundays. Georgean Court, 58 Central Park, West, Cor. 66th Street, New York City. Telephone, 2887 Columbus.

Dr. Charles E. Paine, '03, and bride, spent several days of their honeymon viewing the sights of Washington.

Dr. I. H. McConnell died suddenly on October 18th of acute uraemia. He was visiting physician at the National Homœopathic Dispensary. He graduated from the Southern Homœopathic Medical College in 1904.

The Annual Meeting of the Washington Homœopathic Medical Society will be held on December 10th and 11th.

Dr. George M. Dillow announces removal from 223 West 57th Street to 250 West 57th Street, The Rutland, Broadway and 57th Street. Hours, 10-1 P. M., 5:30-6 P. M. Sundays, 12-1 P. M.

Dr. A. B. Norton, '81, announces the removal of his office on the tenth of December to 30 East 55th Street, corner of Madison Avenue. Hours from eight until one o'clock. Disease of the eye exclusively.

Dr. Wm. E. Rounds, '74, regrets to announce that on December 1st, he will retire from practice for an indefinite period. He takes this opportunity of referring his patients to Dr. William Clark McKnight, of 13 Central Park, West, in whose care he has placed his records covering nearly thirty-five years. Dr. McKnight has been associated with Dr. Rounds for a number of years in his hospital practice.

Dr. G. Herbert Taylor, '04, of Maplewood, N. J., has been appointed health officer in his own town.

Dr. Rolla G. Grant, '79, of Rochester, N. Y., died on November 8, 1909, at his home, suddenly, of angina pectoris, æt. 65. He was beloved by his confreres, brother alumni and patients.

BROOKLYN NOTES.

At the December meeting of the Brooklyn Society Dr. Geo. Hills Iler spoke upon emergencies occurring in the course of ordinary disease, showing clinical charts of various cases. Of special interest was his case of hyperpyrexia in rheumatic fever; a comparatively rare case. The best description of which is given by Fagg, of London, who recites twenty-two cases. This hyperpyrexia occurs in patients from the upper walks of life, usually in young subjects, in very mild cases of rheumatism, and tends to relapse quickly. In the doctor's case the patient had apparently recovered from her rheumatism, when, three days after she was discharged, her temperature went to 103.5°, and in four hours reached 107.5°. The rapid rise of temperature is characteristic of the disease, and is not accompanied by pain. Ice baths constitute the treatment, beginning the bath at 95° and lowering to 60°, with ice on the head, abdomen and spine.

Dr. G. W. Knight, D. D. S., presented two cases of fracture of the jaw under treatment by a simple method, which uses the upper jaw as a splint, fastening the lower jaw to it by means of soft copper wires anchored around the teeth. The proper occlusion of the teeth serves as a landmark, no attention being paid to the line of fracture, but it is necessary to study the way in which the facets of the teeth are worn from use, in determining the proper occlusion. The wires are fastened about the teeth of both jaws and the ends are twisted, remaining in place for four to five weeks. The advantages of this simple method, avoidance of surgical operation, parts always in view, accuracy, requires no special splints, no bandage necessary, no models or impressions necessary.

The thirty-ninth annual commencement of the New York State School for Training Nurses was held at the Young Women's Christian Association on December 7th, eight nurses graduating. This training school was the first organized institution of its kind in the United States, and is connected with the Prospect Heights Hospital. Dr. W. W. Blackman presided, and presented the diplomas

and the faculty prize for highest standing, which was won by Miss Emma L. Collins, who also won the prize presented by Dr. H. C. Allen for highest standing in obstetrics. Addresses were made by the Rev. J. Howard Melish and Dr. Wm. H. Price.

Dr. Pierre A. Banker, '79, one of Elizabeth's oldest and best known practicing physicians, recently elected on the Republican ticket as a member of the Excise Commission, died suddenly at 1 o'clock in the morning of December 2d, at his home, 1156 East Jersey street, of heart trouble, with which he had long been a sufferer.

The doctor retired last night at the usual hour. Soon after midnight he complained of an unusual pain about his heart, and Dr. Milton A. Shangle, whose home is near by, was hastily summoned. Dr. Banker failed to respond to the treatement, and soon passed away.

Dr. Banker was a son of John T. Banker, who was a descendant of John and Priscilla Alden. The doctor was born at Poughkeepsie, N. Y., December 5, 1845. He was educated at the Mt. Pleasant Military Academy at Sing Sing, N. Y., and after graduation there was employed for a time as clerk in a bank.

At the breaking out of the Civil War he enlisted as a private in Company A, Twentieth New York Infantry. He afterwards enlisted in the regular army, in which he served five years. He was in the cavalry service on the plains and in General Custer's regiment. He retired from the service in 1871, with the rank of sergeant major, soon afterward entering upon the study of medicine in the New York Homœopathic College, from which he was graduated in 1879. After a brief practice in New York City, he went to Millbrook, N. Y., and then to Rhinebeck-on-the-Hudson, where he remained ten years. He came to this city from Rhinebeck in 1889.

Dr. Banker was a member of the New York State Homœopathic Medical Society, the New Jersey State Homœopathic Medical Societ, the Union County Medical Society and the American Institute of Homœopathy. For several years he was vice-president of the New Jersey State Homœopathic Society. He was spoken of by his friends as a "good doctor as well as a good fighter, and a man of high intelligence and worth."

For many years Dr. Banker took a deep interest in the Independent Order of Foresters. He was a charter member of Court Colum-

bus, No. 920, of that order, and was its examining physician from its organization, eighteen years ago, until the time of his death. He was also for a time examining physician for both Court W. Childs, No. 1476, and Court Boudinot, No. 1285. In 1895 he was honored by being elected as one of the three delegates from this city to attend the Supreme Council of the order, which was held in England. The other two delegates were Willett T. Bingham and the Rev. George Buckle, former pastor of the Greystone Presbyterian church.

Dr. Banker was also a member of Washington Lodge, No. 33, F. and A. M., and of Garfield Conclave, No. 241, Order of Heptasophs, of this city. He was also a comrade of Ulric Dahlgren Post, No. 25, G. A. R., an organization in which he ever took much interest.

Dr. Banker's wife, to whom he was married on November 13, 1873, was Miss Julia Carroll, daughter of William Carroll, who was a prominent merchant and business man at Rhinebeck. He was an attendant at St. John's church. Besides his wife he is survived by two sons, and two daughters, George T. Banker, the well known organist; Pierre Augustine Banker, Jr., and the Misses Harriet Preston and Julia Carroll Banker. He also leaves one brother and three sisters, W. B. Banke, of Passaic; Mrs. J. B. Derby, of Wayne, Pa., and Mrs. W. W. Emerson, of Brooklyn, N. Y.

ANNOUNCEMENTS.

At the present time there are a few vacancies in the Training School at Flower Hospital. Applications should be addressed to Supt. Training School, Flower Hospital.

The Trustees of Columbia University have appointed Dr. William G. MacCallum, Professor of Pathological Physiology in the Johns Hopkins University, to be Professor of Pathology at Columbia in succession to Dr. T. Mitchell Prudden, who will retire from active service on July 1 next.

Dr. MacCallum is a graduate of the University of Toronto and of Johns Hopkins Medical School. He is regarded as one of the very foremost investigators and teachers in the field of pathology. The list of his original contributions to pathology is not only long but remarkable, by reason of the extent of the field covered by Dr. MacCallum's studies during the past ten years.

At the same time the Trustees have made provision for the development and extension of the Department of Bacteriology under Professor Philip H. Hiss, Jr., and of Clinical Pathology under Professor Francis C. Wood. Increased attention will be paid by all of these departments to the needs of advanced students and investigators, as well as to the instruction given to students of medicine.

A very thorough course on Public Health and Sanitation has been established at the Medical School of Columbia University, and representative scholars and public health officers from all parts of the country are co-operating in the course.

The lecturers include Professor Sedgwick, of the Massachusetts Institute; Professor Adami, of McGill University; Professor Burr, Holt and Peterson, of Columbia; United States Surgeon General Wyman, State Commissioner Porter, Dr. Flexner, Director of the Rockefeller Institute; and the experts of the New York Health Department.

Professor Gies, of the Department of Biological Chemistry, is conducting a course of public lectures on The Chemistry of Digestion, on Monday evenings, at Cooper Union. The lectures will be illustrated by zoetrope figures, and by chemical and digestive demonstrations.

DR. JOHN E. WILSON, President. DR. JAMES E. TYTLER, Secretary.

TRANSACTIONS OF THE ACADEMY OF PATHOLOGICAL SCIENCE.

Regular Meeting, October 22, 1909.

EVIDENCE CONTROVERTING ROSENBERGER'S TUBERCULAR BACTERÆMIA HYPOTHESIS.

By Bond Stow, A. M., M. D.

Pathologist Metropolitan Hospitai, B. I., New York City.

Shortly after Doctor Randle C. Rosenberger, of Philadelphia, advanced his hypothesis that "tuberculosis in whatever form is a bacteræmia," I was fortunate to receive, through the kindness of Dr. Henry G. Piffard, of this city, an invitation to meet Dr. Rosenberg and learn from him his precise technique and the ideas he held in reference to this extremely important subject. My interest was aroused and following that interview, for three months, I devoted all of my time that could be spared from actual practice to a series of investigations along the lines suggested by him with results considerably at variance with those announced by him.

In the September 18, 1909, number of the Journal of the American Medical Association, appeared an article by Dr. Brem, who likewise had not been able to obtain Dr. Rosenberger's results *after modifying the Rosenberger technique,* in which article he pointed out some very interesting sources of error (see end of this report).

In order that one thoroughly may understand the conflicting results it is essential to point out the variations in our technique.

From the American Journal of Medical Sciences of February last I read from Dr. Rosenberger's article, as follows:

Rosenberger's Technique.

"I took about 5 c.c. of blood from a vein of the arm. This was immediately placed in an equal quantity of a 2 per cent. solution of Sodium citrate in normal salt solution. The mixture was well shaken and placed in the refrigerator for twenty-four hours. At the end of this time there was an abundant sediment with the citrate solution slightly turbid. Centrifugalization immediately after mixing did not give very good results so I determined upon letting the mixture stand for twenty-four hours. After this time I pipetted off a quantity of the sediment and made a rather thick preparation upon a new, clean glass slide, dried it upon a copper plate with

moderate heat and then placed it in distilled water until complete laking of the blood resulted. At this time only a delicate film remained. This was dried and fixed through a bunsen flame and then stained by the usual technique for tubercle bacilli. As a rule, bacilli possessing the morphological and tinctural properties of the tubercle bacillus could be demonstrated in the first slide thus prepared, although in several cases three slides were thoroughly searched before any bacilli were found. A negatve result would not be announced until, at least, thirty minutes of careful searching had been given to a slde."

NOTE.—I did not announce a negative result in my work until I had given three solid hours to each slide.

Dr. Rosenberger, in his conclusion, says: "It appears that tuberculosis in all its forms is a bacteræmia" and adds in a footnote that up to January 15th, 1909, he had examined 125 cases and detected tubercle bacilli in all.

I immediately began independent studies along these lines with the following simplified technique, for I believe the more simplified the method of obtaining results the greater the accuracy and the less the liability of adverse criticism.

Procedure in Bedside Cases.

A ligature was placed above the elbow and the arm thoroughly asepticized. The blood taken from any prominent vein was immediately smeared over the entire surface of two slides that previously had been thoroughly cleaned with soapsuds and then placed in the following solution:

	Gms.
$K_2Cr_2O_7$	50
H_2O	200
H_2SO_4	300

They were left in this solution for twenty-four hours or more, then rinsed and immediately before the smear was made, passed two or three times through a flame. The smear was then allowed to dry in the air, then fixed by rapidly passing several times through the flame and stained with carbol-fuchsin and destained with 25 per cent. hydrochloric acid in 95 per cent. alcohol. I did not at any time *lake* the blood. Thus I took care that my slide was as free from contaminations as possible, simply placing the blood on the slide directly from the arm and staining immediately without the use of water or any other substance.

The specimen was now examined direct without the use of a cover glass. I found the acid fast bacilli deeply stained and presenting the usual morphology of similar bacilli as found in sputum examinations. In no case were doubtful or suspicious appearances accepted as positive. On each slide I passed in review hundreds of fields, giving not less than three hours' time in negative cases.

Results.—Ten incipient cases where positive acid-fast bacilli were found in the sputum, showed absence of any acid-fast bacilli in the blood. Eighteen

advanced cases showed acid-alcohol-fast bacilli in the blood in six cases and absence of same in the balance.

Procedure in Autopsy Cases.

Now as to my procedure in autopsy cases. All were cases of undoubted advanced peribronchial type of tuberculosis with cavity formation, in which, in short, there was no question as to tuberculosis. The heart was tied off from the main blood vessels and removed with its blood. The surface of both ventricles was thoroughly seared with a large red-hot knife as well as placed directly in the flame and then the blood aseptically taken from each ventricle and smeared upon the slides with the technique already explained. As to the lungs, they were removed entire and after drying as much as possible were bisected with a knife passed previously through a flame. One of the larger pulmonary veins was then selected and after allowing the first blood to pass off that within the vein was taken with an aseptc platinum spatula and smeared on the slide. Every possible precuation was taken not to touch any of the adjcining tissues and the absence of extraneous bacteria on my slides would go to prove the freedom from contaminations displayed in my work.

Now as to the result of eleven autopsy cases in which I took the blood from the right and left pulmonary veins and both ventricles of the heart.

CASE NO.	LT. LUNG	RT. LUNG.	LT, VENTRICLE	RT. VENTRICLE
1	+	O	O	O
2	+ (4 bacilli in all)	O	O	O
3	+	+	O	O
4	+	+	O	+
5	O	+ (one bacillus)	O	O
6	+	O	O	O
7	+	+	O	O
8	+	+	O	O
9	+	+	O	+
10	+ (Large numbers of Phagocytes)	+	+ Sparce	+ Sparce
11	O	O	O	O

The results of the above experiments are at variance with those of Rosenberger and *Forsythe . . . who both used the same methods which differs from mine in that they *laked* the blood before staining. Now what

*British Medical Journal, April, 1909.

bearing has the laking of the blood upon their results? Dr. Walter V. Brem in Vol. 53, No. 12, of the Journal of the American Medical Associaton appears to give the answer. Brem found that there was unmistakable contamination of distilled water with acid-fast bacilli in laboratories where animals are kept, and you will obtain constantly positive results before elimination of this source of contamination as long as you lake the blood. Brem says: "Rosenberger fails to state explicitly whether or not a thorough search for acid-fast bacilli in the citrate and oxalate solutions and in the distilled water used in laking the blood was made by fixing the sediment with an albuminous material. This procedure in technique is the crux of the whole matter. "One result of his anmal experiments, thirty guinea pigs showing acid-fast bacilli in the blood specimens while having no tuberculous lesions is strong evidence in favor of contamination rather than the presence of tubercle bacilli in the circulating blood."

In my work no distilled or tap water was used before staining. This is the way I account for the difference between the results of Doctors Rosenberger and Forsythe and myself.

Certainly this whole problem is a very difficult and momentous one, and it seems to me there must be more uniformity in results before Rosenberger's hypothesis that "tuberculosis in all its forms is a bacteræmia" can be raised to the position of a demonstrated fact. There is no doubt that tubercle bacilli do occur at times and under certain conditions in the blood in cases of tuberculosis but the When and How have not, as yet, definitely been establshed. We are not in a position yet to state positively that every case of tuberculosis is a case of bacteræmia.

I have brought with me a microscopical specimen of phagocytosis taken from the blood of a case of tuberculosis in which we see very distinctly nine acid-alcohol-fast bacilli being phagocyted—if I may coin the word—by a polynuclear leucocyte. The stain happens to be very clear and distinct.

[The specimen was examined, with great interest, by the members present.]

DR. AVERY. in response to the invitation of the chair, said he could add very little to what had been stated except that, after Dr. Rosenberger published his work on the subject he and Dr. White had undertaken a line of research similar to that followed by Dr. Stow. They had followed Dr. Rosenberger's technique very carefully and accurately and in 50 cases taken from the tubercular wards of Bellevue and Kings County Hospitals, cases in which the clinical diagnosis of tuberculosis was umistakable, the slides were absolutely negative. In no case did they find acid-fast bacilli or bacilli of any sort; and animals inoculated and autopsied at the end of six or eight weeks showed no suspicion of any lesion of tuberculosis.

DR. LAIDLAW said he had been watching Dr. Stow's work since early spring, but did not take any personal hand in it. He visited Dr. Rosenberger's laboratory to see if he used anything that we did not have here, as he was reporting positive results in Philadelphia. Dr. Rosenberger very kindly took him through his wards and brought a specimen of sputum

from the ward to his laboratory. He laked the slide, staining it by a method suggested by Dr. Laidlaw and in about ten minutes after the staining was completed had bacilli under the microscope that could not be distinguished from acid-fast tubercle bacilli.

Dr. Laidlaw had taken 5 c.c. specimens of blood from eight different cases. He treated the first four by Dr. Rosenberger's method and the last four by the antiformin-ligroin method. In none of the eight cases was he able to find the tubercle bacillus in the blood and they were all advanced cases. He had stained several slides and devoted fully one hour to each slide.

He believed that one German observer had recently used the antiformin method in an attempt to demonstrate tubercle bacilli in the blood and his results corresponded pretty well with those of Dr. Stow. In the last stages of the disease he found them in about 40 per cent. of the cases; in advanced cases in about 20 per cent. and in incipient cases not at all, while in four cases of joint tuberculosis he found the bacilli in the blood of all of them. If tuberculosis were, in all its forms, a bacteræmia and we could find the bacillus in the blood it would be an advance in diagnosis of the greatest importance; but as to the practical value of Dr. Rosenberger's method he felt obliged to confess a large degree of skepticism.

DR. MILLS had attended the meeting of the National Association in Washington in May last, where Dr. Rosenberger read a paper supplemental to what he had already published. There must have been a hundred people present at the session at which the paper was read and among them some fifteen or twenty pathologists and tuberculosis specialists, who reported that they had followed Dr. Rosenberger's methods as nearly as possible with negative results. One man reported that out of 41 cases he had found the tubercle bacillus in the blood of not more than three. They assailed not only his findings, but his technique, from start to finish. He got very angry before the meeting came to an end. One man from Bellevue said they had gotten Dr. Rosenberger to come to New York and Dr. Rosenberger could take his subject, get his specimen and find the tubercle bacilli and the next man taking the same subject could not find them. They did not know how he did it and they could not find out.

DR. PIFFARD said that when he first heard of Dr. Rosenberger's method he went to his laboratory and saw him practicing his technique. When he came home Dr. Stow brought some blood specimens to his laboratory and Dr. Lefevre did the same. They worked hard over the case and utterly failed. Dr. Rosenberger sent him a slide prepared from human blood and one also from bovine blood, in which there were frequent fields containing acid-fast bacilli, which certainly, if found in ordinary clinical cases, would have been accepted as the tubercle bacillus; but whether they were or not he did not know.

The story from Bellevue was not quite accurately reported to Dr. Mills. Dr. Rosenberger was invited to come to Brooklyn to demonstrate on New York patients his method. The laboratory there was in cahrge of Dr. White

and Dr. Avery. The blood was taken from patients whom Dr. Rosenberger approved. The slides were prepared on Saturday and examined on Sunday following. He had the privilege of being present. They examined possibly half a dozen specimens from different tuberculous patients. Dr. Rosenberger did not find, nor even pretend to find, a single tubercle bacillus. Neither did Dr. White nor Dr. Avery. He had never seen a more nearly broken-hearted man in his life than Dr. Rosenberger, who could not understand it. He found them in Philadelphia but he could not find them in New York. None of us in New York had found them except very occasionally and then, possibly, under extenuating circumstances [laughter]. What could possibly be the explanation? He could conceive of none until last summer he was so fortunate as to meet Dr. Brem, of Colon, and Dr. Brem told him of the experiments he had made in which he had found the bacillus in the blood and that after finding it he had gone carefully over his technique and found that it was the distilled water that held the acid-fast bacilli.

Dr. Piffard had been told that Dr. Rosenwein, of Washington, had had a similar experience with the water. They could find them in the water of Philadelphia, Colon and Washington. He remembered reading in a paper some two or three years ago that acid-fast tubercle bacilli had been found in the Schuylkill water in Philadelphia. Apparently New York water is free from acid-fast. He had never known acid-fast to turn up unexpectedly where they could not be accounted for. Indeed, the acid-fast bacilli are pretty extensive. It is well known that Muller, in 1889, first announced that he had found acid-fast in timothy grass which so closely resembled the tubercle bacilli as to be distinguished from them only with difficulty. Dr. Piffard had several cultures made from timothy and last July gathered some timothy grass up in Herkimer county, planted them in sterile water and within a few days the tubes were filled with acid-fast bacilli, the most perfect imitation of the bovine bacillus that could possibly be imagined. And, furthermore, Dr. Avery had told him that cultures made this year exhibited bacilli of the human type, there being quite a difference between the appearance of the human bacillus and the bovine type.

DR. SAYRE reported thirty cases tried at the Metropolitan Hospital. These cases he had for convenience, divided into five classes.

The first class, under the ordinary method, showed an average of one bacillus to a field, after the use of antiformin-ligroin, 12 to the field.

Class II., by usual methods, averaged 1 to 2 in 5 fields; after antiformin-ligroin, 70 to the field.

Class III., showed 1 bacillus to 10 to 25 fields and after antiformin-ligroin, 9 to the field.

Class IV., showed 2 bacilli to the slide; after antiformin-ligroin, 9 to the field.

Class V. were negative to ordinary methods of these; after antiformin-ligroin, 7 were negative, 4 positive. The first of the 4 positive showed 6 to the field, the second, 4 to 25 fields and the other two 1 to 25 fields.

THE ANTIFORMIN LIGROIN METHOD FOR FINDING TUBERCLE BACILL WHEN SCANTY.

By Dr. George F. Laidlaw.

Mr. President and Members of the Academy:

I wish to introduce a new method of examining sputum, fæces and all material containing tubercle bacilli; its peculiar advantages being that it isolates and concentrates the bacilli. In the diagnosis of tuberculosis we must admit that the one sure diagnostic point is the finding of the bacilli. We have the X-ray and the percussion note and the Kroenig method of estimating the width of the apex, which I consider very valuable, but the X-ray is difficult to apply and the percussion note may be misleading. The finding of the bacillus is the one sure sign. Therefore any method which enables us to find the bacillus more easily or to find it earlier in the history of the case than is now customary must become a very valuable method in diagnosis.

Such a method is this which I wish to present to you this evening.

Antiformin is a liquid resembling ordinary Javelle water with the addition of strong soda lye. It is an intense oxidizer and has the peculiar property of dissolving organic substances. The formula was patented in 1900 by a Swedish engineer and was first employed in breweries for cleaning pipes. Uhlenhuth used it first in dissolving sputum and found that it not only dissolved the mucus but also killed and dissolved bacteria. Staphylococci, streptococci, typhoid bacilli, gonnococci and all the rest of them, with one exception, disappeared in a fifteen or twenty per cent. solution of this material. In treating sputum he found one type of bacterium that did not disappear, the acid-fast bacteria, of which the tubercle bacillus is the most prominent representative He could mix up a sputum containing half a dozen different kinds of bacteria and at the end of five minutes every bacillus in the mixture was killed except the tubercle bacillus. Anthrax spores are also quite resistant. He realized that here was a simple method of isolating the tubercle bacillus. He devised a technique of mixing the sputum with antiformin, spreading it in a Petri dish and fishing out the lumps for examination or centrifuging and examining the sediment. Lange and Nitsche first made use of the ligroin method and Bernhardt combined the two. Ligroin is a light hydrocarbon resembling benzin. It is of lighter specific gravity than water and has the property, which is possessed by all light oils, that, if introduced into an emulsion containing tubercle bacilli and shaken thoroughly, the little drops of oil will attach themselves to the bacilli and carry them to the top.

I have here a dram of sputum which was declared negative by the usual method. I examined the patient and diagnosed fibroid phthisis. The antiformin-ligroin method easily showed groups of bacilli.

Make a twenty per cent. antiformin solution (I recommend making the solution at the time you use it. I think there is little loss of strength if

kept for any length of time. Anywhere from fifteen to thirty per cent. solution is effective). To 5 c.c. of sputum in a cylindrical graduate or ordinary two-ounce bottle, add 20 c.c. of the 20 per cent. antiformin, put in the stopper and shake thoroughly. The more it is shaken the better. In about twenty minutes most sputa are liquified. Sometimes there are viscid masses that take longer than that to dissolve. They dissolve more qucikly in an incubator. The rate of solution depends upon the strength of the antiformin and the frequency of shaking. I have been letting the more viscid specimens stand over night. When dissolved, dilute the specimen with a little water to reduce its specific gravity, pour about one-eighth of an inch of ligroin on top and shake again vigorously. Emulsifying the oil reduces it to very minute drops. The tubercle bacilli adhere to these drops and rise with them to the top. resting at the junction of the antiformin and the oil. Then take out half a dozen loopfuls, place on a slide and stain in the usual manner. There is another point in the technique worthy of note. The loopful of benzin evaporates very rapidly on a warm slide and so you can keep putting one loopful on top of another, concentrating a number of drops on one spot. To your surprise you may find a sputum in which, at first, you failed to find any bacilli, reveal with this method fifteen, twenty or fifty in a field. Personally, I can vouch for the great value of this method in the examination of sputum.

Furthermore, the antiformin does not seem in any way to affect the vitality of the bacillus. Bacilli that have been treated with twenty per cent. antiformin grow well on culture media and are virulent for animals. Ordinarily, isolating tubercle bacilli from sputum is difficult because the contaminating bacteria grow so rapidly as to cover over the slowly growing tubercle bacilli. With antiformin, the other bacteria are killed, leaving the tubercle bacilli to grow uncontaminated. Fifty per cent. antiformin solutions kill the tubercle bacilli, too.

Another point of value is that any pathological material, as urine or fæces (which it deodorzes), or parts of an organ as a piece of a kidney or a lymphatic gland can be treated in the same way and dissolved, leaving the bacilli free. You can see the advantage over the old metohd of hardening, cutting a dozen sections, staining, mounting and then hunting for hours for your bacillus.

This leads to another phase of the subject, the difficulty in finding tubercle bacilli in undoubtedly tubercular tissue; and its explanation by the form of the tubercle bacillus that does not stain with Ziehl. This discovery was made in von Behring's laboratory by Dr. Much.

Dr. Much inoculated several calves with a culture of tubercle bacilli. When killed, they were found distinctly tubercular, but in the specimens of tissue from one animal examined he could not find a single tubercle bacillus. In trying a variety of stains he devised one which revealed the bacilli in great numbers. In addition, he found heaps of fine granules, some arranged in forms simulating bacilli and others like heaps of dust. On staining pure cultures of tubercle bacilli in the same way, similar heaps of fine granules were found.

This observation seems to explain several things. It explains why it is that when you send scrofulous glands and other tissue to the microscopist they fail to find any bacilli in them. Dr. Sayre will show a section of tubercular kidney in which no bacilli were demonstrable by Ziehl but in which delicate blue bacilli are revealed in the tubercles by the Much stain.

This observation of Much may explain another curious thing which I have observed but have not been able to explain.

In tubercular phthisis, the bacilli sometimes disappear from the sputum, and then, for some unaccountable reason, they will come back. Schulz, of Davos, investigated ten of these cases with the Much method and found the dotted bacilli, the granules and the dust-like heaps.

Now, while the Much stain is useful for caseous glands and cold abscess fluid which usually contain no other bacteria, sputum and urine contain many other bacteria which stain with the Much stain and this confuses the result. It has been suggested that antiformin will dissolve all other bacteria so that a combination of antiformin and the Much stain would be specific for the tubercle bacilli. While antiformin dissolves most bacteria, I have observed a few remaining cocci and bacilli which were not tubercle bacilli after one week in antiformin. They were scanty, but still the presence of only a few undissolved bacilli would vitiate the results.

It is probable that a method by antiformin isolation and culture would give better results. The idea deserves further development and improvement in technique.

Dr. Dieffenbach asked whether the antiformin method might not be open to the same objection which seemed to be pretty well established against Dr. Rosenberger's method; contamination of the water used in the process, which might contain acid-fast bacilli.

Dr. Laidlaw replied that the method did not dissolve any acid-fast bacteria. It seems to be the acid-fastness which protects them. Smegma bacilli and timothy bacilli would not be dissolved. In his own experience he had not found tubercle bacilli where there was no reason to expect them to be present or where there was not good clinical evidence of tuberculosis. But on the whole it must be admitted that there might be contaminatoin of the water unless freshly distilled water were used.

Dr. Sloat had nothing to add to what had been said by Doctor Laidlaw except that he had used the method referred to and found it very useful. The thing that impressed him most was the very rare occurrence of negative sputum where there was any clinical evidence of the presence of tubercle bacilli. He had taken sputum from Metropolitan and Flower Hospitals which had been reported negative and very often had found them positive after prolonged search. The point is that a sputum cannot be called negative until one has spent, at least, an hour on the slide using a mechanical stage and bringing several hundred fields into view.

Dr. Piffard thought that ligroin was probably not the only oily substance that could be used for the purpose. It is well known that any ordinary oily

substance like milk seems to have a sort of chemotaxis for the various bacilli. His attention had been drawn to this fact some two years ago by a chemist who told him how to get rid of the bacteria in beer, which are sometimes troublesome. The chemist would pour his fluid into the beer vat and stir it up and after a while he would skim off the bugs from the top. He wanted a good price for his discovery and they were willing to pay it, when presently they found out that he was using ordinary sweet oil. There is unquestionably an attraction between certain bacteria and various fats which perhaps explains the fact that ligroin holds them, it being a hydrocarbon.

Dr. Laidlaw further stated that he wished to correct any possible misapprehension that might have occurred in reference to ligroin. It was preferred only because of its specific gravity. Ordinary benzin will do the work also. He himself had started with petroleum, ether and xylol. Almost any light oil would bring the bacilli to the top, but it must not rise too quickly and thus tear itself away from the bacilli.